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A. EXPERIMENTAL ANIMAL DECOMPRESSIONS TO A NEAR-VACUUM ENVIRONMENT.

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B. EXPERIMENTAL ANIMAL DECOMPRESSIONS TO LESS THAN 2 MM. HG ABSOLUTE (PATHOLOGIC EFFECTS)

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FOREWORD

These reports were prepared in the Physiology Branch under task No. 775801. The study was supported by contract No. DPR T-16758-G with the National Aeronautics and Space Administration, Manned Spacecraft Center, Houston, Tex. The papers were presented at the Aerospace Medical Association Meeting, Miami Beach, Fla., on 12 May 1964 and submitted for publication on 9 April 1965.

Experiments reported herein were conducted according to the "Principles of Laboratory Animal Care" of the National Society for Medical Research.

The valuable technical assistance of Major William D. Habluetzel is most gratefully acknowledged.

This report has been reviewed and is approved.

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ABSTRACT

To estimate the times of consciousness, collapse, and survival of animals exposed to near-vacuum environments, 126 conscious dogs were rapidly decompressed in either 1 or 0.2 second from 35,000 feet, while breathing oxygen, to a pressure less than 2 mm. Hg absolute. Groups of 6 dogs each were exposed to this low pressure for periods of time ranging from 5 to 180 seconds, with and without prior denitrogenation, and then recompressed to 35,000 feet with oxygen in either 5 or 30 seconds. The dogs collapsed within 9 to 10 seconds after decompression, as determined from motion picture films. Simultaneously, the effects of anoxia, water vapor, and other evolved gases were apparent, resulting in a generalized muscle spasticity, a few gasps, momentary convulsive seizures, apnea, and gross swelling of the body and extremities. All dogs exposed for less than 120 seconds survived, despite evidence of lung involvement. Respiration recommenced spontaneously either during recompression or at ground level, provided the heart was beating; otherwise, death was inevitable. The longer the exposure time, the more prolonged was the time for recovery which usually ranged from a few minutes to a few hours, except for 1 dog which exhibited a severe postdecompression paralysis with gradual recovery over a period of several weeks. Exposures of 120 to 180 seconds resulted in approximately 15% to more than 80% fatalities, respectively. Denitrogenated dogs tended to show a slightly better survival rate. As might be expected, the shorter the exposure time and the faster the recompression rate with oxygen, the better were the chances for uneventful and prompt recovery.

EXPERIMENTAL ANIMAL DECOMPRESSIONS TO A NEAR-VACUUM ENVIRONMENT

I. INTRODUCTION

Despite the fact that a considerable number of studies have been carried out on the effects of rapid decompression to high altitudes, there is still very little information and data concerning the actual effects of exposures to extremely low barometric pressures—that is, to pressure environments approaching the near-vacuum of space. This information is becoming increasingly urgent in view of the current manned space flights, the programed flights to the surface of the moon, and the need for man to function safely within a pressure suit in space.

The extensive series of studies carried out by Hitchcock and his co-workers (5), as well as by other investigators (2, 3, 4, 6), have defined quite clearly most of the effects of rapid decompressions to at least 30 mm. Hg absolute, using experimental animals. Whether or not rapid exposures to even lower ambient pressures approaching that of a vacuum result in even more profound consequences cannot be entirely deduced from this earlier work. Some questions that are still not clear, but must be answered with a reasonable degree of confidence, are concerned with:

1. Time of consciousness.

2. Survival time after loss of consciousness.

3. Extent and type of pathologic effects that might occur in the vital organs.

4. The biologic effects of interstitial and intravascular water vapor.

5. The effect of denitrogenation on the time of consciousness, survival, recovery, and residual effects.

The critical situation confronting an aerospace crew should accidental loss of pressure be experienced dictated the use of physiologically normal animals so that the data collected would be as valid as was possible to obtain. Normal, unanesthetized dogs were therefore used; 126 animals were rapidly decompressed to absolute pressures of 1 to 2 mm. Hg.

II. METHODS

The experimental procedures and decompression-recompression profiles are summarized in table I.

In most tests, 3 dogs were simultaneously decompressed. The rapid decompressions were all from 35,000 feet (180 mm. Hg) with the chamber flooded with oxygen. In this way, the animals remained unrestrained and hypoxia was avoided at this altitude before the decompressions. No animal was decompressed more than once.

TABLE I

Experimental procedure

All decompressions from 35,000 feet (in oxygen)		
Decompression times:	1 second	(108)
	0.2 second	(18)
Duration of exposures (6 animals in each group) 3-5, 10, 30, 60, 90, 120, 135, 150, 165, 180 seconds		
Recompression times:	5 seconds	(84)
	30 seconds	(27)
Denitrogenation: 1 hour at ground level		(42)
Type of recompression gas:	Oxygen	(117)
	Room air	(9)

Numbers in parentheses are number of animals.

Decompression times from 180 to less than 2 mm. Hg were all approximately 1 second, except for 6 faster decompressions (18 dogs) which were arranged to occur within approximately 0.2 second.

Exposure times at the absolute pressure of 1 to 2 mm. Hg for the various groups of animals ranged between 4 and 180 seconds. At least 2 groups of 3 each were exposed to this low pressure for approximately 4, 10, 30, 60, 90, 120, 135, 150, 165, and 180 seconds. In this way the critical times for the onset of unconsciousness, survival, recovery, and nonsurvivability were estimated from the effects on the 6 animals for each exposure time and for each variation in the experimental conditions—i.e., rate of recompression and time for denitrogenation before decompression.

Recompression times to 35,000 feet (180 mm. Hg) with pure oxygen were 5 seconds (28 decompressions, 84 dogs) and 30 seconds (9 decompressions, 27 dogs), followed within 10 to 15 seconds by recompression to ground level with room air. For comparison, 3 additional decompressions (9 dogs) were carried out using air instead of oxygen during 5-second recompressions.

To determine whether or not denitrogenation before decompression provided any degree of protection, 42 dogs (14 decompressions) breathed a high concentration of oxygen ($> 90\%$) for 1 hour at ground level in the decompression chamber before the rapid exposures to low pressure. Decompression times, exposure times, and recompression times for these denitrogenated animals were similar to those already described.

The time course and chamber pressure changes for each decompression were recorded through a Statham pressure transducer on a Honeywell Visicorder. From these records, the time for each decompression could be accurately determined, as well as the exposure times and the recompression times. In addition, motion pictures were obtained for a large number of the decompressions, with an appropriate timer and pressure gage visible in the pictures. With these film records, the precise times for complete unconsciousness and collapse could be determined, as well as the time required for the extensive swelling due to water vapor and other evolved gases. Exposure times, recompression times, and the barometric pressure at which the emphysematous swelling tended to deflate and return to normal during recompression were also determined from these film records.

To establish, as well as possible, uncomplicated baselines for survivability and recovery, no artificial respiration or other methods of resuscitation were used on any of the animals after recompression to ground level pressures. Provided the pulmonary airways are open, the process of recompression itself is as effective as a deep inspiration for ventilating the lungs, regardless of whether or not the animals are breathing. Undoubtedly, any resuscitation efforts, including the continuous administration of oxygen and possibly "overcompression" in a high pressure chamber, would have been of additional benefit during the process of recovery.

III. RESULTS AND DISCUSSION

Survivability, tolerance, and recovery from these severe exposures to extremely low pressures were better than had at first been anticipated, particularly in view of the rapid onset of virtually complete anoxia, together with the boiling effect and gas evolvement in the body fluids and tissues.

Most of the observations that were made at this extremely low pressure coincided in many respects with those previously reported by Edelmann and Hitchcock (2) for dogs exposed to 30 mm. Hg absolute. The hemorrhagic lesions found in these earlier investigations can, in part, be attributed to the extremely rapid decompression rate, reported as 0.03 second, from virtually ground level pressures (750 mm. Hg) (2) and from 522 mm. Hg to 87 mm. Hg in 0.012 second (3). The decompression phase in the present study was much less severe in terms of rate and pressure change (a pressure

change of approximately 180 mm. Hg in 1 second), even though the final pressure reached was much lower (approximately 1 mm. Hg absolute).

Time of consciousness and collapse

All animals exposed to the low pressure for longer than 5 seconds tended to lose consciousness and began to swell and collapse within 9 to 11 seconds. For brief exposures of only 4 to 5 seconds with recompression to 180 mm. Hg on pure oxygen, definite indications of collapse were exhibited by most of the animals at about the 11th or 12th second after the start of the decompression, even though recompression was in progress or had been completed. Under these conditions, however, with 5-second recompressions, postural unsteadiness was transitory when it occurred, and apparent recovery with signs of conscious orientation was rapid. When the recompression time to 180 mm. Hg with oxygen was prolonged to 30 seconds, after 5-second exposures, a more profound state of collapse occurred at about the 10th second, with recovery commencing during the recompression at about 140 mm. Hg (40,000 feet). These time relationships are shown in table II.

The exact moment when loss of "useful" consciousness commenced was difficult to determine merely by observation, but it must certainly have begun to occur before actual postural collapse. Thus, consciousness very probably began to fade at the 8th or 9th second, if not sooner, at least for those animals exposed to the low pressure for longer than 5 seconds.

TABLE II

Onset of postural collapse after rapid decompression from 35,000 feet to approximately 1 mm. Hg absolute

Exposure time at 1 mm. Hg	Recompression time	Collapse occurred within
	(seconds)	
< 5	5	11 - 12
< 5	30	10
> 5	5	9 - 11
> 5	30	9 - 11

With regard to human exposures to a vacuum under similar conditions, it is suggested from these results that, for minimal loss of consciousness, recompression with oxygen to at least 180 mm. Hg (35,000 feet) must begin within 5 seconds and must be completed within the next 5 seconds (although these times cannot be stated with certainty). Figure 1 shows a comparative extrapolation for times of consciousness at altitudes above 55,000 feet, with less than 9 to 10 seconds being cautiously estimated for exposure to a vacuum on the basis of the collapse times measured in dogs. The time of "useful" consciousness appears to be less than the 12 to 15 seconds that have been measured in human subjects breathing oxygen at 50,000 to 55,000 feet (7).

Water vapor effect and subcutaneous swelling after decompression

During the decompressions all animals showed essentially the same response. Immediately after decompression, the animals exhibited excitation and increased activity for the first 5 seconds. Within the next 5 seconds, unless the animals were promptly recompressed, they began to show marked evidence of gas expansion and water vapor evolvement. This was manifested by a high degree of subcutaneous emphysema and expulsion of gas from the stomach and lower bowel, often with simultaneous projectile vomiting, defecation, and urination similar to that previously reported by others (2, 6). The water vapor effect and gas expansion were of such magnitude that the animals became completely immobilized with the extremities, neck, and body in an extended position, similar in appearance to an inflated goat-skin bag. Oddly enough, the external ears and the eyeballs did not seem to show the effects of this phenomenon and remained essentially normal in appearance, although the soft tissue around the eyes and face

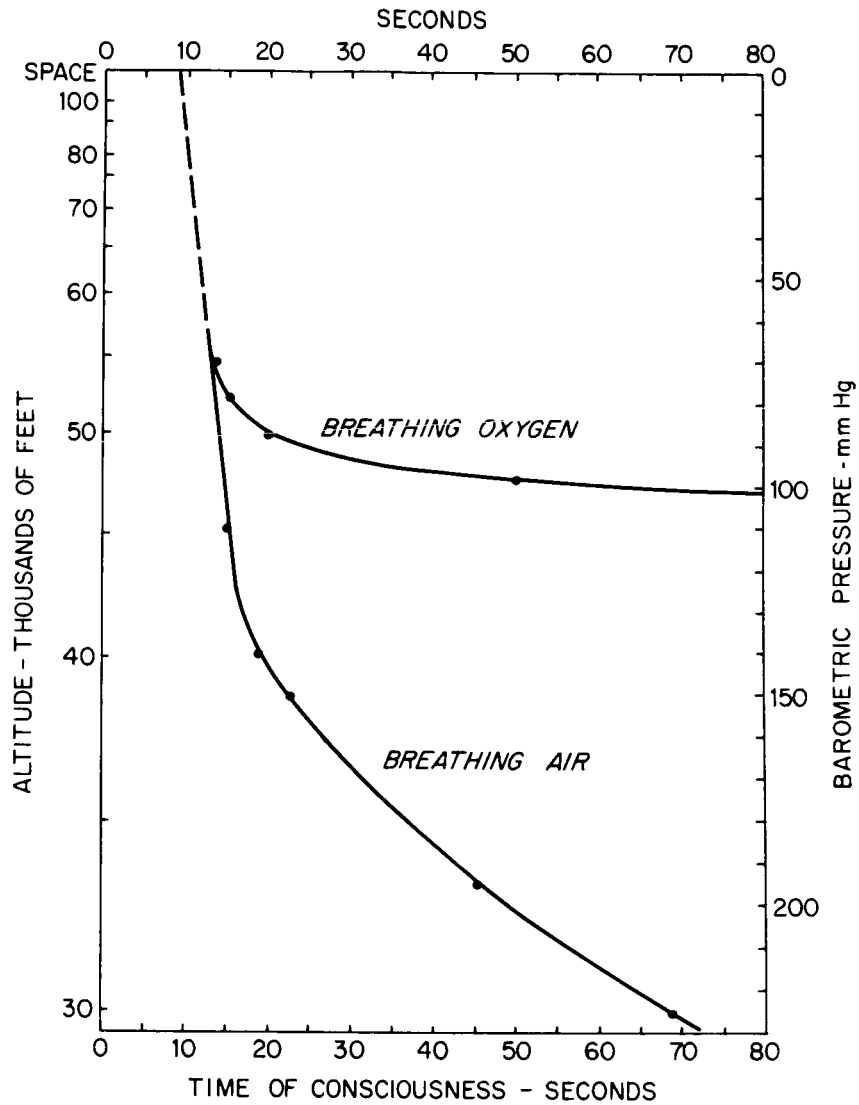

FIGURE 1

Time of consciousness for human rapid decompressions to 55,000 feet (7) with a tentative extrapolation (dashed line) to lower pressures based on canine data obtained at less than 2 mm. Hg absolute.

was often grossly distended, as was the tongue. By the end of the first 10 seconds, as indicated above, all the animals appeared to be unconscious. This was followed by what seemed to be a *grand mal* convulsion, ending in apnea and a spastic rigidity which progressed to a flaccid paralysis, although the animals remained grossly distended. All the animals had entered into this state of flaccid paralysis within about 30 seconds and remained so until they were recompressed. While at the low pressure, the saliva-like secretions and the urine became frozen and partially dehydrated. It was also noticed in several animals, after recompression to ground level, that the tongue was coated with ice. This was particularly true for animals exposed to the low pressure for longer than 2 minutes when evaporative cooling from these exposed wet surfaces became increasingly effective.

Recompression and recovery

During recompression to higher pressures, the subcutaneous gases were, of course, also recompressed and the animals quickly and dramatically deflated to their normal appearance, still remaining, however, in an obvious state of flaccid paralysis, unconsciousness, and apnea. This deflation process appeared to begin rather gradually with the onset of recompression. At 25 to 30 mm. Hg absolute, deflation became more rapid; at approximately 70 mm. Hg the animals appeared to have returned to their normal size. At 45 to 50 mm. Hg, however, a major portion of the deflation is complete, suggesting that water vapor is probably the predominant gas concerned with the excessive distention of the animals. The exact pressures at which the deflation process and the condensation of water vapor occurred was influenced, in part at least, by the subcutaneous and deep body temperatures which, in turn, were probably affected by the duration of the low pressure exposures and the evaporative cooling of the body surfaces.

The rapidity of recovery during or after recompression was generally dependent on the duration of the low pressure exposure, the rate of recompression, and on whether or not the animals were recompressed with oxygen or air. As might well be expected, the shorter the exposure time and the faster the recompression with oxygen, the more rapid and less complicated was the recovery period. Animals that were exposed to the low pressure for 90 seconds or less often began to breathe spontaneously during the recompression to ground level. When the exposures to the reduced pressure were longer than 90 seconds, the depressed state of the animals was intensified and apnea persisted for a prolonged period of time after recompression. Under these conditions, when first examined at ground level, the animals were usually apneic with variations in heart rate ranging from bradycardia to tachycardia. They remained apneic for varying periods of time, but spontaneous respiration always began in less than 2 to 3 minutes, provided there was a heartbeat. Otherwise, when no heartbeat was detectable, the animals invariably failed to survive. During the course of recovery, both the heart rate and respiratory frequency increased steadily for the first 2 to 5 minutes. Some of the animals exhibited a state of decerebrate spasticity when stimulated by being touched or handled. Most of the animals started purposeful movements of the extremities and head within 10 to 15 minutes and next progressed to a stage showing disorientation, with staggering and apparent blindness. During this time, coordinated control and strength in the hind legs seemed to return much more slowly than in the forelimbs. By the end of 30 minutes, none of the animals exhibited objective neurologic abnormalities; nevertheless, they appeared to be in a state of extreme fatigue and exhaustion and were very lethargic. The apparent blindness seemed to abate by the end of the 30th minute of recovery. Those that were exposed to the low pressure for longer than 60 seconds excreted, after recompression to ground level, an excessively large amount of clear, saliva-like fluid from the mouth; moist, basal rales were audible, suggesting pulmonary edema. By the end of 24 hours, the animals spontaneously cleared themselves of the rales and edema and appeared to have normal respiratory function and behavior.

Mortality incidence

As mentioned above, all dogs exposed to the near-vacuum environment for less than 120 seconds survived with essentially uneventful recoveries despite evidence of severe but transitory lung involvement. On the other hand, exposure times ranging from 120 to 180 seconds resulted in mortality rates of about 15% to more than 80%, respectively, as shown in figure 2. Each symbol in this figure represents a group of 6 animals from which the percent mortality was computed. It can be noted that no deaths resulted from the 90-second exposures, regardless of whether the animals were recompressed (with oxygen) to 35,000 feet (180 mm. Hg) within 5 seconds or 30 seconds. The 120-second exposures, however, resulted in 1 death (16.7%) in the 6 animals that were recompressed in 5 seconds and 3 deaths (50%) in the group of 6 animals that were recompressed in 30 seconds.

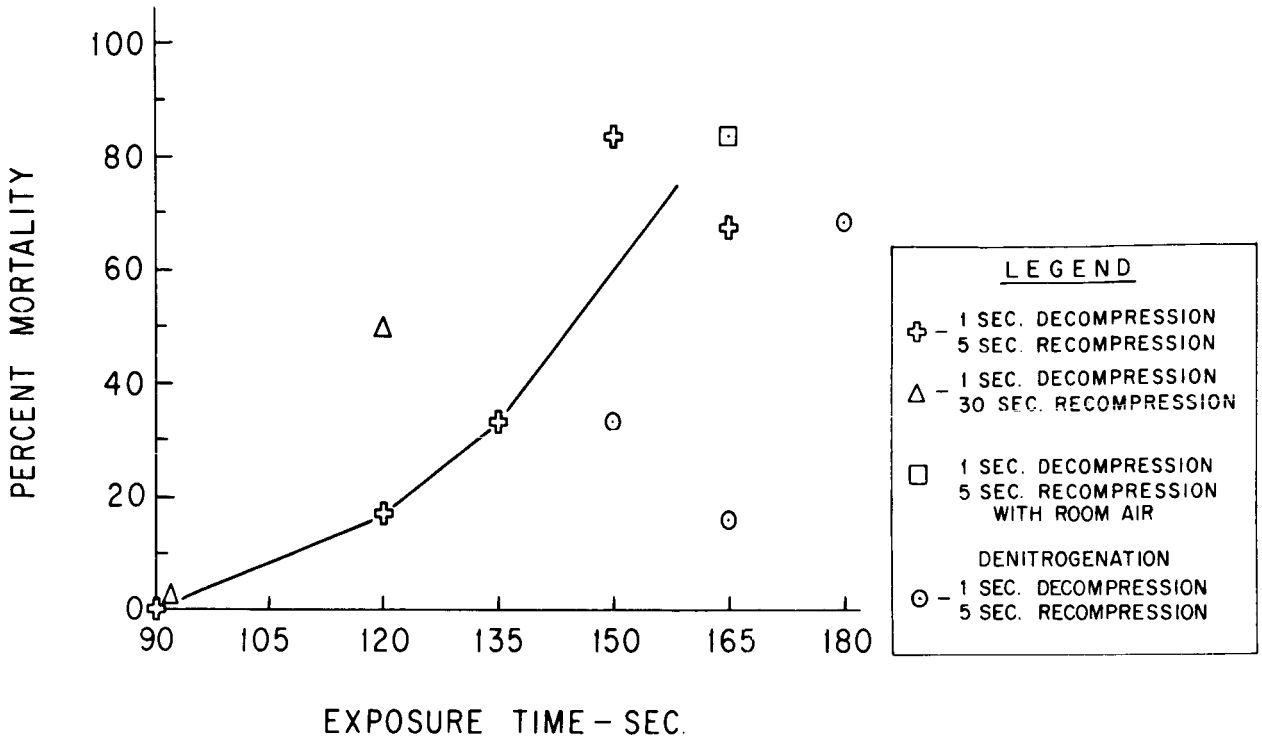

FIGURE 2

Percent mortality resulting from rapid decompression exposures to less than 2 mm. Hg absolute with and without prior denitrogenation and with 5- and 30-second recompressions to 180 mm. Hg absolute with and without oxygen as noted. Each symbol represents a group of 6 dogs. The curve for 5-second recompressions (with oxygen) was approximated by eye.

Of the 9 dogs that were rapidly recompressed with room air rather than oxygen, 3 were exposed to the low pressure for 150 seconds and all survived. The other 6 animals were exposed for 165 seconds and only 1 survived (83.3% mortality.) These results, with only a limited number of animals, indicate that the mortality incidence is not particularly different from that of animals exposed for the same period of time and then recompressed with oxygen. There are sound reasons to believe, however, that recompression with oxygen provides for more rapid oxygenation of the blood at a lower pressure than when room air is used (8). As mentioned above, recompression from near-vacuum conditions, regardless of whether or not the animals are breathing, is as effective as a deep voluntary inspiration. If oxygen is used as the recompressing gas, it virtually assures that the lungs are filled with oxygen, provided the airways are open. On the other hand, when room air is used, it is necessary to recompress virtually to ground level pressures (750 mm. Hg) for adequate oxygenation of the lungs and blood, equivalent to 180 mm. Hg (35,000 feet) when oxygen is used.

Dogs that were denitrogenated for 1 hour at ground level before decompression and then recompressed with oxygen (fig. 2) had a significantly lower mortality rate ($P < .05$) as a result of these prolonged severe exposures. With denitrogenation, 5 out of 6 dogs survived a 165-second exposure, and 2 out of 6 survived an exposure of 180 seconds. It may be postulated that the cerebral and other vital tissues and fluids, when completely denitrogenated and fully saturated with oxygen before decompression, permit an extra margin of

several seconds after decompression before the intracellular oxygen partial pressure decreases to profound anoxic levels. This is possibly reflected in the longer survival time compared to the survival time of animals that were not as completely denitrogenated.

In general, under these conditions, the shorter the exposure time and the faster the recompression rate with oxygen, the better are the chances for survival and uncomplicated recovery (excluding the possibility of middle ear blockage during recompression).

Observations made on small primates

In connection with another study carried out by Rumbaugh and Ternes (10), 20 trained squirrel monkeys (4 groups of 5 each) were also decompressed in the same manner as described above, except that no exposures to the low pressure were longer than 90 seconds.

After decompression to approximately 1 mm. Hg absolute, the squirrel monkeys appeared to lose consciousness sooner than the dogs. As with dogs, they had both tonic and clonic seizures shortly after unconsciousness and this progressed to flaccid paralysis. Subcutaneous emphysema and swelling occurred, but was not as marked. During and following recompression to ground level, the monkeys recovered similarly but seemed to exhibit staggering and disorientation for a longer period.

Two monkeys died as a result of these low pressure exposures, while no dogs died from exposures that were less than 120 seconds. The first death occurred after a 10- to 12-second exposure with a 30-second recompression to 35,000 feet (180 mm. Hg) with oxygen. This animal, contrary to that seen in nonsurviving dogs, had a perceptible but irregular heartbeat after reaching ground level, but never recovered spontaneous respiration. Necropsy revealed apparent basalar atelectasis of the lungs with a questionable perforated visceral pleura just over an area of petechial hemorrhage.

The second fatality resulted from a 90-second exposure with a 5-second recompression to 35,000 feet. This monkey exhibited no heartbeat and no spontaneous respiration after reaching ground level. Except for pulmonary atelectasis and a few subpleural petechial hemorrhages, no gross pathologic abnormalities were observed at autopsy, which was performed under water.

Residual postdecompression paralysis and other nervous disorders

In only 1 animal was there clear-cut evidence of residual central nervous system involvement that continued to persist for 24 or more hours after recovery from prolonged exposure to the low pressure. The fact that approximately one-third of the surviving animals were sacrificed and autopsied within 30 minutes postdecompression leaves open to question whether or not this small percentage (approximately 1%) might not be actually larger had all survivors been observed for at least several days before sacrifice. It seems reasonable that the probability of residual pathologic conditions in the central nervous system would be increased considerably with increased exposure times, particularly for prolonged exposures of 2 minutes or longer when the chance for survival itself becomes marginal.

The one dog that exhibited severe postdecompression paralysis had been exposed to the low pressure environment for 120 seconds and then recompressed with oxygen to 180 mm. Hg in 30 seconds. After reaching ground level, the dog reacted in much the same manner as the other animals in the group, with a bradycardia and apnea which were quickly resolved. This animal, however, manifested prolonged visual impairment, and 1 hour after the exposure had not completely regained vision, as tested by reaction to motions of objects. There were no other overly abnormal neurologic manifestations at this time compared to the other animals similarly exposed. When seen approximately 20 hours later, this animal was unable to stand, but appeared to be spirited and mentally alert. Neurologic examination showed that the extremities on the right side were weak, with depressed deep tendon reflexes; while extremities on the left side were spastic, with hyperactive reflexes. The cranial nerve and pain

response reflexes were present, and the animal seemed to have unimpaired sensation to pinprick. There was also spastic urinary incontinence. Vision appeared to be normal. During the following days, progressive improvement occurred so that by the end of 1 week, the animal could walk unassisted, although the extremities on the left side still showed considerable impairment. At this time, a urinary tract infection developed without fever, but was easily controlled with antibiotics. During the next 12 weeks, the neurologic condition improved steadily; at the end of this time, the animal was able to walk and run well. The hemiparesis was scarcely discernible by neurologic examination, and the spasticity of the left side and bladder was absent.

Inasmuch as this animal had been subjected to a 120-second exposure with a 30-second recompression, the chances of survival were marginal. For a group of 6 or more animals under these same conditions, about 50% fatalities could be expected (fig. 2). Thus, in many instances, animals which otherwise might show evidence of considerable damage to the central nervous system probably do not survive. Those that do survive show, for the most part, relatively uneventful recoveries. Only in a few rare cases where the threshold of death has been very closely approached is it possible to demonstrate survival with clear-cut residual and, possibly, irreversible lesions of the central nervous system. Human hypoxic incidents in aircraft (9) and low pressure experiments with animals, by Büchner (1) and others cited by him, suggest similar conclusions.

From these observations on dogs and small primates, conclusions concerning survivability and recovery indicate that the prevention of death by rescue and repressurization with oxygen within the order of 90 seconds appears to be feasible. The fact that a fatality from a 10-second exposure has been observed in a small primate proves that any exposure to such low pressures involves great risk, and there can be considerable individual variations. Some dogs have survived exposures for as long as 3 minutes with apparently good recovery, whereas one dog suffered severe but nonfatal damage to the central nervous system after an exposure of 2 minutes.

REFERENCES

1. Büchner, F. Pathology of general oxygen deficiency. *In* German aviation medicine World War II, vol. I, ch. IV-O, p. 419. Washington, D.C.: U.S. Government Printing Office, 1950.

2. Edelmann, A., and F. A. Hitchcock. Observations on dogs exposed to an ambient pressure of 30 mm. Hg. J. Appl. Physiol. 4:807 (1952).

3. Edelmann, A., W. V. Whitehorn, A. Lein, and F. A. Hitchcock. Pathological lesions produced by explosive decompression. J. Aviation Med. 17:596 (1946).

4. Gelfan, S., and A. Y. Werner. Cardiovascular responses following explosive decompression of macaque monkeys to extreme altitudes. J. Appl. Physiol. 4:280 (1951).

5. Hitchcock, F. A. Studies in explosive decompression. WADC-TR 53-191, Wright-Patterson AFB, Ohio, 1953.

6. Kovalenke, Ye. A. The problem of the mechanism of development of high-altitude tissue emphysema. *In* Sisakyan (ed.). Problems of space biology, vol. 2, p. 407. USSR, 1962.

7. Luft, U. C., H. G. Clamann, and E. Opitz. The latency of hypoxia on exposure to altitude above 50,000 feet. J. Aviation Med. 22:117 (1951).

8. Luft, U. C., H. G. Clamann, and H. F. Adler. Alveolar gases in rapid decompression to high altitude. J. Appl. Physiol. 2:37 (1949).

9. Luft, U. C. Altitude sickness. *In* Armstrong, H. G. (ed.). Aerospace medicine, ch. 9, p. 139. Baltimore: Williams and Wilkins, 1961.

10. Rumbaugh, D. M., and J. W. Ternes. Learning set performance of squirrel monkeys after rapid decompression to vacuum. Aerospace Med. 36:8 (1965).

B. EXPERIMENTAL ANIMAL DECOMPRESSIONS TO LESS THAN 2 MM. HG ABSOLUTE (PATHOLOGIC EFFECTS)

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FOREWORD

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Experiments reported herein were conducted according to the "Principles of Laboratory Animal Care" of the National Society for Medical Research.

The technical assistance of Technical Sergeant Joe Rawdon, Staff Sergeant Jesse Vasys, and Airman First Class John C. Lingafelter is gratefully acknowledged.

This report has been reviewed and is approved.

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ABSTRACT

Pathologic examination of tissues of dogs rapidly decompressed to less than 2 mm. Hg absolute was performed. Of the 126 dogs decompressed, 92 were autopsied at 3 time intervals: within 30 minutes, 2 to 5 days, and 1 to 3 weeks postdecompression. Gross examination of the tissues was performed on all autopsied animals. Lung damage was graded 1+ to 4+ according to the amount of edema, emphysema, atelectasis, and hemorrhage present alone or in combination. Microscopic examination of the tissues was performed on selected dogs from the various groups.

The most impressive finding was the absence of major pathologic damage, except in the lungs, unless the exposure time exceeded 120 seconds. By varying time of decompression and time of exposure to less than 2 mm. Hg, it was possible to separate the pathologic effects of anoxia versus time of decompression. In all dogs, the severity of lung damage increased with duration of the anoxic exposure. In groups with comparable exposure times, the dogs decompressed in 1 second exhibited pulmonary congestion, edema, and hemorrhage, while those decompressed in 0.2 second showed predominantly more petechiae and emphysematous changes. Denitrogenation appeared to reduce the incidence and severity of the lung damage. Animals autopsied at the later postdecompression periods showed evidence of resolution of all lesions, especially in the lungs. For the exposures that were longer than 120 seconds, gross examination of other organs and tissues showed increasing congestion and some hemorrhage. The brains showed engorgement without evidence of hemorrhage. One dog that was paralyzed from the exposure had numerous demyelinated lesions of the spinal cord that seemed to be the result of gas bubble emboli.

Author

EXPERIMENTAL ANIMAL DECOMPRESSIONS TO LESS THAN 2 MM. HG ABSOLUTE (PATHOLOGIC EFFECTS)

I. INTRODUCTION

In the period 1946 to 1953, with the advent of the pressurized cabin, numerous studies were reported on "explosive" decompressions. The studies enumerated the pathologic effects resulting from decompressions ranging from ground level to 72,000 feet (30 mm. Hg) in 0.030 to 0.080 second (11). Because of recent exploits into space and the consequent hazard of being exposed to a vacuum, it is necessary to determine the effects of near-vacuum pressures (less than 2 mm. Hg) and the morbidity inflicted from lethal and sublethal exposures to this atmosphere. Denitrogenation, oxygen or air recompression, and slow or fast recompression could influence the pathologic damage resulting from a rapid decompression. The present decompressions were designed to elucidate the pathologic abnormalities that could be anticipated with 0.2-second or 1.0-second decompressions from 35,000 feet (180 mm. Hg) to less than 2 mm. Hg for varying periods of time with or without denitrogenation and recompressed in 5 or 30 seconds with 100% oxygen or room air. From the variables used and the material available from past experiments, it is possible to separate some of the causative factors responsible for the tissue damage in rapid decompressions to near-vacuum pressures.

II. METHODS

Of the 126 dogs that were decompressed, 93 were autopsied, and specimens were taken from 76 for fixation. The 29 dogs that died and 23 of the survivors that were sacrificed were autopsied within 30 minutes post mortem. Of the remaining survivors, 27 were sacrificed and autopsied 1 to 6 days postdecompression, and 13 were autopsied 7 to 21 days postdecompression, except for 1 dog (G-74), which was sacrificed on the 87th day after decompression. The time of sacrifice was predetermined so that from each group of 3 dogs decompressed at the same time, 1 of the dogs was sacrificed within 30 minutes, another in 1 to 6 days, and the last in 7 to 21 days after the decompression. All the survivors were anesthetized with pentobarbital and then perfused with 10% formalin through a catheter placed in the left ventricle of the heart. All organs were examined grossly and sections of the brain, lungs, heart, liver, kidneys, adrenals, spleen, gastrointestinal tract, gonads, skin, muscle, and bone were taken for microscopic examinations in representative cases. All tissues, except the brain, were microscopically examined in 22 dogs and, among these, 12 brains were examined. Of the 8 spinal cords that were removed, 5 were sectioned at intervals of 3 to 4 cm. for microscopic examination. Three dogs, that had not been decompressed, served as controls and were sacrificed and autopsied in the same manner as the others. The tissues of all the animals were embedded in paraffin for hematoxylin-eosin staining or, in some cases, frozen for fat staining. The brains and spinal cords were stained with cresyl violet, Weil's myelin, and Van Gieson's solution. Holzer's crystal violet stain for gliosis was used on selected brain and spinal cord sections.

III. RESULTS

Because of the complexity of the experiments and the large number of animals investigated, the pathologic effects will be considered by organ systems. Although the

amount of damage to any of the organ systems was small in relation to the magnitude of the insult, the findings of most significance were in the lungs, kidneys, liver, and central nervous system in certain cases.

Lungs

Gross examination of the lungs revealed intra-alveolar edema, subpleural petechiae, or ecchymoses, regional atelectasis, peripheral emphysematous changes, and hemorrhagic atelectasis. These findings were subjectively graded by one investigator according to the following scale:

+ = Mild edema, emphysema, or petechiae, separately.

++ = Any two of the above, moderate edema (significant), atelectasis, marked emphysema, or numerous petechiae.

+++ = Severe edema, marked atelectasis, numerous ecchymoses, or less than 30% hemorrhagic atelectasis.

++++ = Greater than 30% hemorrhagic atelectasis.

Figure 1 shows the lung damage incurred as a result of a 1-second decompression and 5-second recompression with varying times at 1 mm. Hg. The severity of the lung damage increased with the duration of exposure. Small petechiae, areas of emphysema, and atelectasis were predominant in the shorter exposures. In the longer exposures (longer than 120 seconds) pulmonary edema, ecchymoses, and patches of hemorrhagic atelectasis were predominant, whereas little of the former type of damage was seen. The other two columns in each exposure time are representative of animals that survived and were sacrificed 1 to 6 days postdecompression and 7 to 21 days postdecompression. Animals sacrificed at later dates demonstrated that the survivors had reversible lung damage because resolution was underway in all cases.

It is worthy of note that exposures of less than 90 seconds to the near-vacuum pressures resulted in mild lung damage (grade + or ++) while exposures for longer than 90 seconds induced serious lung damage (grade +++ or ++++). The upper lobes seemed to be most susceptible to emphysematous changes which

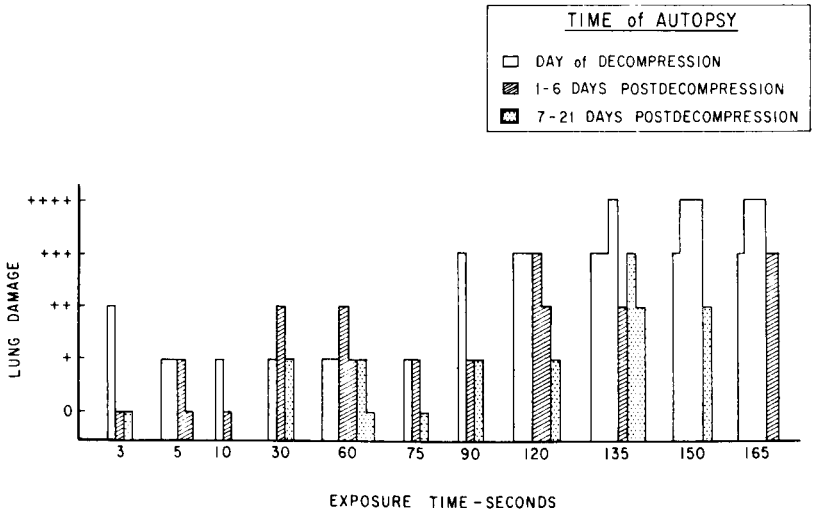

FIGURE 1

Rapid decompressions from 180 mm. Hg to less than 2 mm. Hg in 1 second with 5-second recompressions. Each column represents 1 dog. The times are the duration at peak altitude.

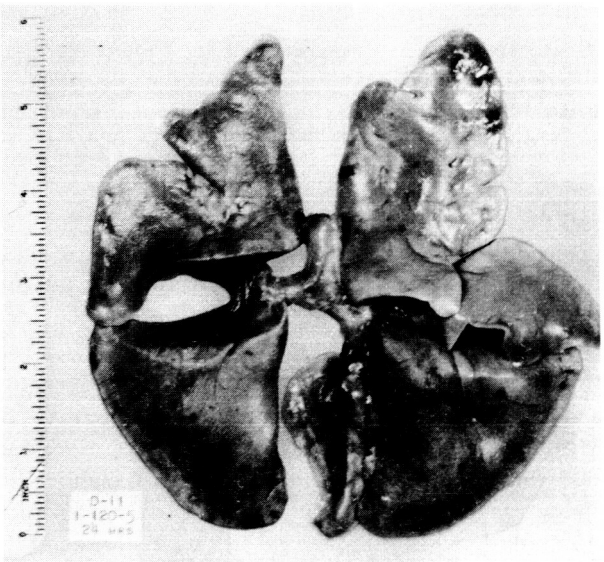

FIGURE 2

One-second decompression to less than 2 mm. Hg for 120 seconds and recompressed in 5 seconds. Dog sacrificed 24 hours later. Note numerous scattered subpleural petechiae with mild edema of basal lobes and minimal peripheral emphysema of left upper lobe. (Grade ++)

were usually peripherally distributed. Edematous and atelectatic findings were found predominantly in the lower lobes basally. The petechiae and ecchymoses were randomly distributed (figs. 2, 3, and 4). Vascular congestion was increasingly prominent in the longer exposures, especially those that were longer than 120 seconds. Microscopic examination of lung tissues from selected animals confirmed the gross findings. A chronic resolving pneumonitis with patchy infiltration of plasma cells and lymphocytes and occasional microabscesses were seen in 2 dogs examined microscopically.

Generally, in the faster decompressions (0.2 second), subpleural petechiae were more abundant. Animals recompressed in 30 seconds had lung damage comparable to extending the time of exposure at vacuum (1 to 2 mm. Hg) by 15 seconds. Denitrogenation exercised a protective effect on the extent of lung involvement so that the animals exposed for 150 and 165 seconds had 2+ to 3+ graded damage, while the dogs which were similarly exposed without denitrogenation had 3+ to 4+ graded damage. Recompression with air or 100%

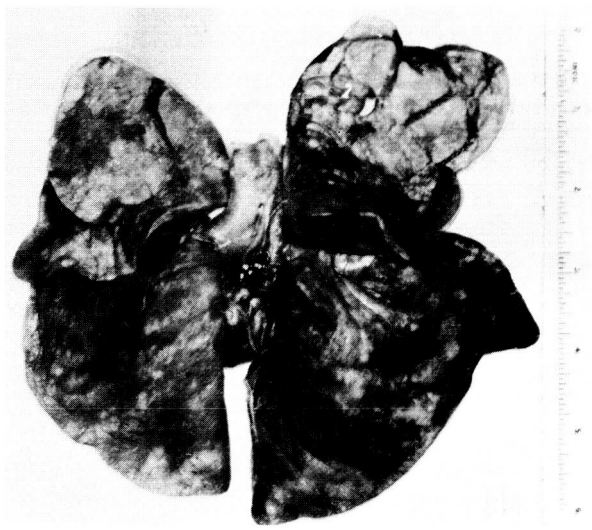

FIGURE 3

Death after 1-second decompression, exposed for 135 seconds and recompressed in 5 seconds. Note more pronounced apical emphysema and large ecchymoses with basal edema and atelectasis. (Grade +++)

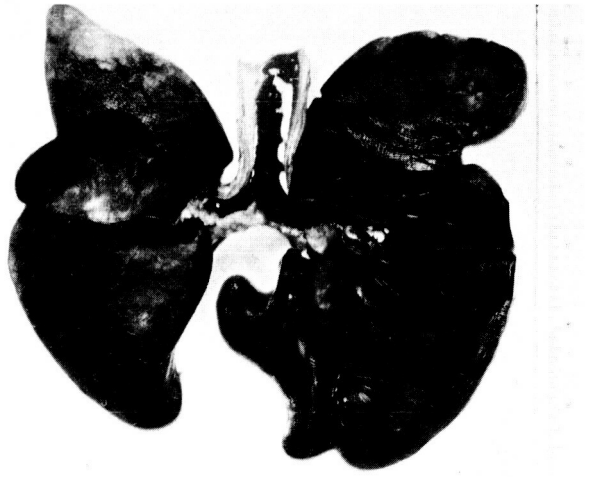

FIGURE 4

Death after 1-second decompression, 150-second exposure, and 5-second recompression. Note massive hemorrhagic atelectasis with hemorrhagic exudate in tracheobronchial tree.

oxygen made little difference. The lungs of the control dogs showed only mild, passive congestion, and in 2 of these there was an occasional microscopic collapsed alveolus.

Liver

Gross examination of the livers revealed no abnormalities except acute, passive congestion in animals exposed for more than 120 seconds. This also was evident in the kidneys, brain, gastrointestinal tract, and spleen. The longer the exposure, the more marked was the congestion, so that dogs exposed for 180 seconds had severe congestion. This was more notable in dogs that died than in survivors that were sacrified within a few hours postdecompression.

Microscopic examination of the liver of 6 of the animals, which died or were sacrificed within 24 hours after exposure, demonstrated diffuse vacuolization of the cytoplasm of the parenchymal cells (fig. 5). These were determined not to be fat deposits. The amount of vacuolization increased with the duration of exposure. These changes were not seen in the animals sacrified at later dates. The variations in time of decompression, rate of recompression, and denitrogenation had no discernible effect on the liver.

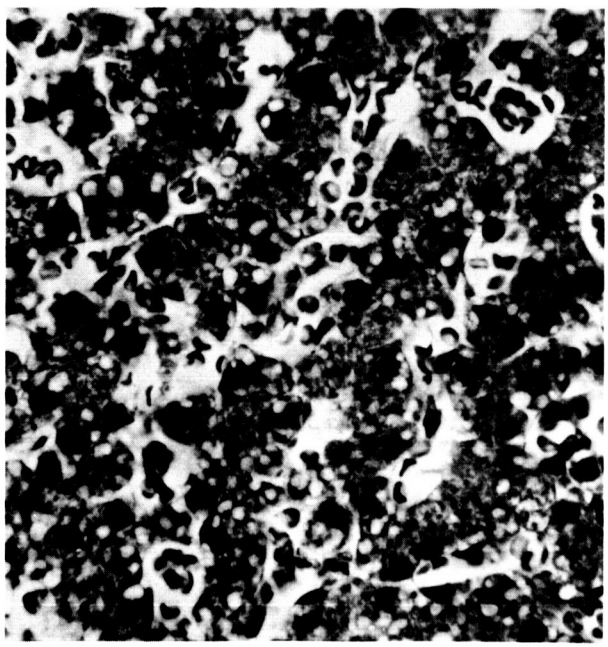

FIGURE 5

Liver of dog sacrificed within 24 hours after exposure. Cytoplasmic vacuolization of parenchymal cells H and E (450×).

Kidneys

Gross examination of the kidneys was unremarkable and there appeared to be no findings related to the decompressions.

Tissue sections of 3 of the selected cases which died or were sacrified within 24 hours showed deposition of some granular yellowish-brown pigment in the cytoplasm of the tubular epithelial cells. This finding was not seen in the sections of the control dogs or those animals sacrificed at later dates. All 3 of these animals were exposed for 120 seconds or more. Bilirubin, iron, and PFAS, acid-fast, and 72-hour oil-red-O stains were negative. The pigment refracted under fluorescent light and gave a positive Schmorl's reaction. This combination of reactions places the pigment in the category of oxidized lipofuchsin. Variations in time of decompression, rate of recompression, and denitrogenation caused no notable differences.

In these tests we found no evidence of increased red blood cell fragility and hemolysis, as noted by Booth (1) in dogs breathing 5% oxygen in nitrogen.

Brain

Gross examination of the brains revealed vascular engorgement in most of the brains of animals which survived decompression for 120 seconds or longer. No other changes were observed externally or on cross section.

Microscopically, the few changes encountered were minimal. In 1 dog there were conspicuous foci of tissue sponginess around occasional larger veins in the thalamus and the rostral-most part of the basic pendunculi (figs. 6 and 7). The spongy tissue contained no coagulated fluid. In the thalamus, the nerve cells and glia included in the focus were shrunken, but there was no glial response, while in

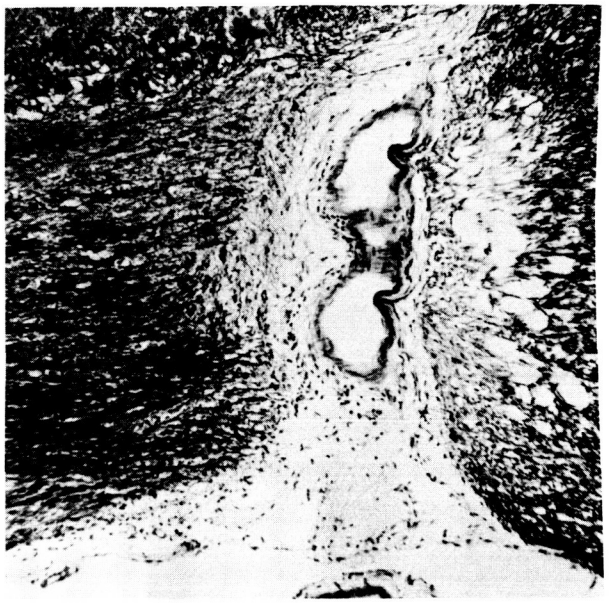

FIGURE 6

(Dog D-25) Rostral-most part of basis pedunculi. Tissue around vein is spongy and demyelinated. Some enlarged glial nuclei are to be seen in the spongy tissue. Mononuclear cells are present in the vascular sheath and vicinity. Weil myelin sheath stain (81×).

the perivenous focus in the basic pendunculi the tissue was demyelinated, glial activation was evident, and mononuclear cells of varied type were noted in the sheath of the vein. A few scattered areas of subpial sponginess were noted in the superficial cortex of 2 dogs that were exposed for longer than 120 seconds and sacrificed within 30 minutes after the exposure.

Structures particularly apt to be affected under conditions of hypoxic hypoxidosis, such as the cerebral cortex, the thalamus, Sommer's sector of the hippocampus and the Purkinje-cell layer of the cerebellum were doubly checked, but no lesions were found. Also, "ischemic" lesions in cerebral and cerebellar cortex and white matter, such as have been encountered in decompression sickness (8), were totally lacking.

Spinal cord

In 3 of the 5 spinal cords examined, no changes were observed. In the other 2, the gray matter and, occasionally, the bordering white matter, contained a considerable number of tiny hemorrhages situated perivascularly. This was at cervical and thoracic levels of the cord. There was no glial reaction in the vicinity of the hemorrhages.

Dog G-74 (1-second decompression, 120 seconds at 1 mm. Hg pressure, and 30-seconds recompression) developed a transient paralysis. The brain and spinal cord were sectioned every 0.5 cm. and stained as the others. The spinal cord was severely damaged. Lesions were limited to the white matter and were found in practically every segment of the cord except the sacral segments (figs. 8 and 9). These lesions tended to be most severe at cervical and upper thoracic levels, and the pattern of involvement varied widely from segment to segment. At some levels, the lesions predominated in the posterior columns, and at other levels, in a lateral or anterior column. Usually, the lesions at a given level were multiple. Some were focal and some were diffuse. In the posterior columns, there were

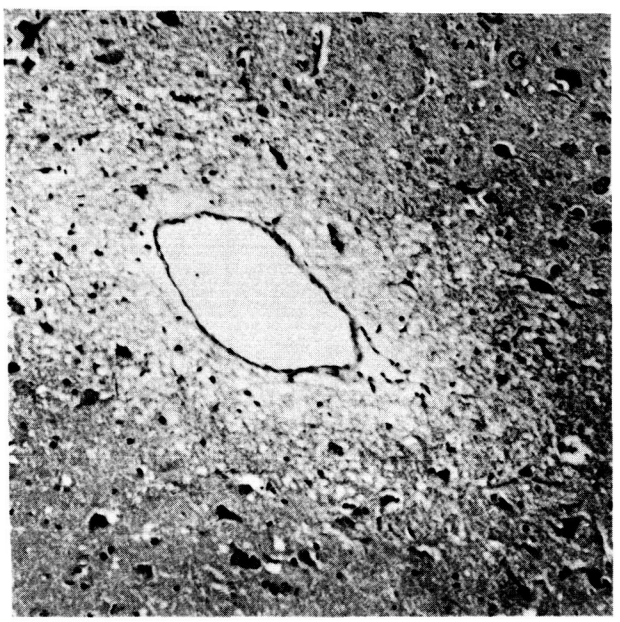

FIGURE 7

(Dog D-25) Thalamus, showing sponginess of tissue around vein. Glia and nerve cells in spongy area are shrunken. Cresyl violet stain (99×).

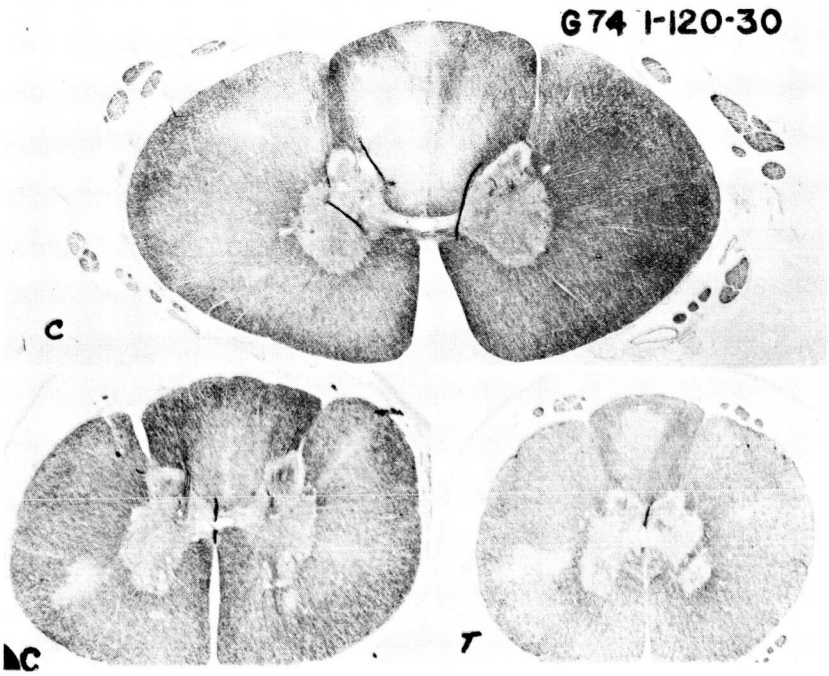

FIGURE 8

(Dog G-74) Lesions in white matter of cord at cervical and thoracic levels. The lesions are multiple and vary in site at the 3 levels. Focal lesions are seen at all 3 levels. Weil myelin sheath stain.

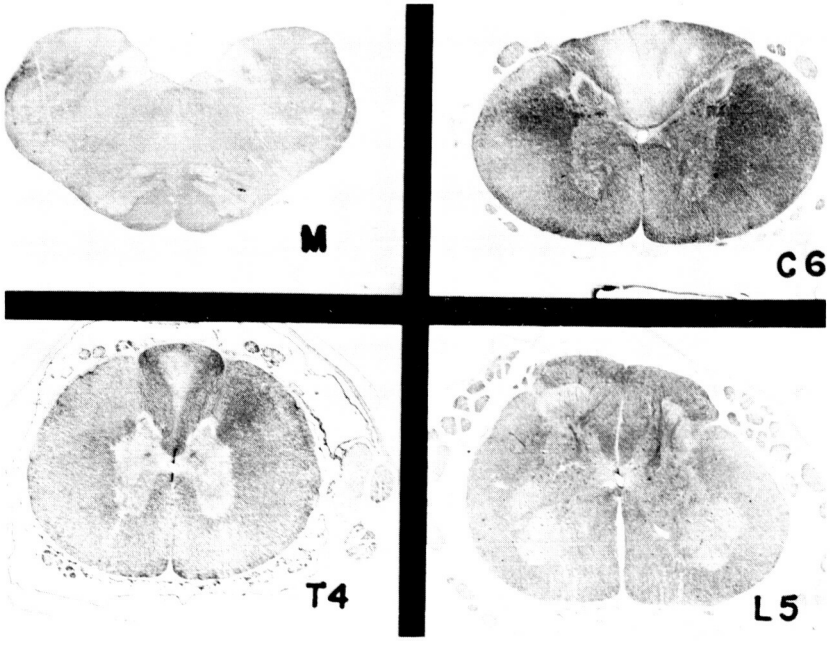

FIGURE 9

(Dog G-74) Medulla oblongata with uniform and symmetric demyelination of fasciculus gracilis and cuneatus bilaterally. Posterior column lesions at cervical of thoracic levels. Note absence of lesions in lumbar section. Weil myelin sheath stain.

solitary lesions which were bilaterally symmetric, while in the lateral and anterior columns the lesions were invariably asymmetric on the two sides of the cord. The lesions predominated in the midregion of the white matter and frequently were wedge-shaped. Rarely did they occupy the region bordering the gray matter. Usually, lesions extended to the pia mater, especially those in the lateral columns. The lesions in the posterior columns had apices centered on the pia near a penetrating vessel and thus had vascular orientation.

The lesions varied greatly in severity. Sometimes, little more than focal myelin pallor was encountered. Characteristically, either in focal or in diffuse lesions, axis cylinders were remarkably enlarged and myelin sheaths correspondingly were dilated or fragmented (figs. 10 and 11). "Vacuoles," which were numerous, were found where axis cylinders had vanished. In most lesions, macrophages and assorted, unidentified, smaller, reactive cells were noted, and in areas of more severe lesions

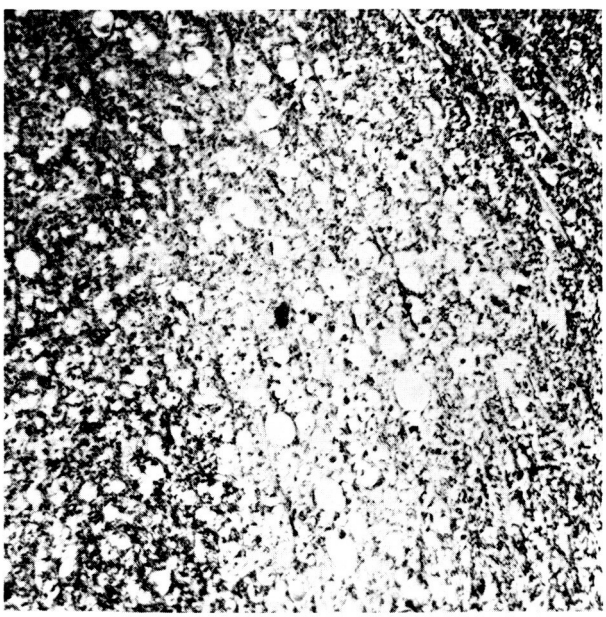

FIGURE 10

(Dog G-74) Lateral column of thoracic segment of spinal cord. The focus shows myelin fragmentation and loss. A strikingly enlarged axis axon cylinder is to be seen. The "vacuoles" represent areas from which individual axis cylinders have vanished. Weil myelin sheath stain (125×).

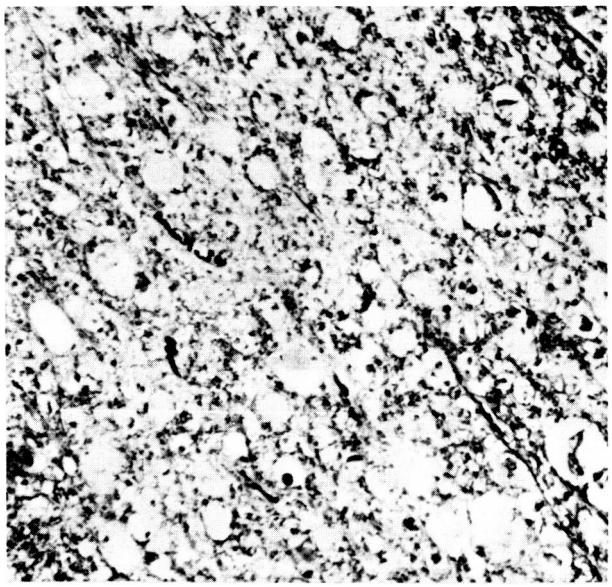

FIGURE 11

(Dog G-74) From severe lesion in posterior column at cervical level. Tissue sponginess is striking. One enlarged axis cylinder is visible, as are also a few enlarged astroglia. Small mononuclear cells are present in vascular sheath. The numerous, widely distributed small mononuclear cells are mostly neuroglia, some of which appear enlarged. Cresyl violet stain (250×).

sparse activated histiocytes and lymphocytes were noted in vascular sheaths. In most of the lesions the oil-red-O stain revealed vast amounts of fat, either in macrophages or free in the tissue, but most abundant along the walls of vessels. Holzer preparations revealed slight astrogliosis in some of the lesions.

Variations in rate of decompression and recompression and denitrogenation caused no effects in the central nervous system. As a result of the decompressions, the other tissues examined showed no significant alteration in structure, not already mentioned, except for dilatation of the right atrium and ventricle; the dilatation was most marked in the fatalities and in dogs exposed for longer than 120 seconds.

IV. DISCUSSION

Although the decompression schedules did produce the anticipated pathologic effects, the most astounding result was the paucity of findings in the animals exposed to such a large

insult. Animals exposed for 90 seconds or less escaped with little damage, and the animals subjected to this environment for 120 to 150 seconds had a good chance of recovery with reversible damage. This is far longer than would have been expected from past studies. Cole et al. (4) decompressed 18 dogs from 10,000 feet to 72,000 feet (500 to 30 mm. Hg) in 0.035 second. The animals were divided into 3 groups: The first group was held at 72,000 feet for 1 minute, and the second and third groups were immediately recompressed after reaching the same altitude. The first and second groups were recompressed in 1 minute, while the third was recompressed at free-fall rate (approximately 7 minutes). Hemorrhages were found in the heart, intestine, stomach, liver, spleen, kidney, and brain, with cell rupture and extrusion of cytoplasm and nuclei from the renal and liver epithelial cells. Fragmentation of myocardial fibers was also noted. Since the authors (4) found no differences in the 3 groups, they concluded that the lesions were a result of the rapid decompression—not anoxia. They also concluded that the fragmentation of the myocardial fibers was the result of a shock wave or of intracellular gas formation.

Our studies were carried out with slower rates of decompression (0.2 and 1.0 second) to a much lower pressure (less than 2 mm. Hg), and with shorter times of recompression (5 and 30 seconds). We were able to demonstrate variation in the extent of pulmonary damage which coincided with the duration of exposure to low pressure. Although our experiments lacked the hemorrhages found in the other organs as well as fragmentation and cellular disruption, the distribution of the pulmonary lesions and mortality were in agreement. In our faster decompressions (0.2 second), petechial and emphysematous changes in the lungs were more frequent. These studies indicate that lesions readily attributable to rapid pressure changes would include petechiae or small-vessel rupture, emphysematous changes or alveolar-septal rupture, and cellular fragmentation and disruption. More extensive hemorrhage, pulmonary edema, and atelectasis seem more related to the duration of anoxia. Additional control studies performed on dogs exposed to 100% nitrogen for periods up to 3 minutes have demonstrated pulmonary edema, atelectasis, and a few parenchymal ecchymoses. These findings lend support to the above conclusion. Atelectatic changes are a common finding in the canine pulmonary system at necropsy.

The protection afforded by oxygen prebreathing (denitrogenation) has been reported (6). The mechanism presumably is increased saturation of body tissues with oxygen so that a longer period of anoxia can be tolerated.

Myocardial cellular fragmentation was not seen in our studies and, therefore, must not be the result of intracellular bubble formation. Rather, it is most likely caused by "shock-wave" formation in extremely rapid pressure changes, as suggested by Cole et al. (4).

The lipofuchsin pigment found in the liver and kidney in the early postdecompression period has not been reported before in other studies of this type. Lipofuchsin apparently represents one of the stages in oxidation of lipid or lipoprotein and the significance of its presence in the tubular epithelium and liver is speculative. However, it must represent an abnormal or increased lipid or lipoprotein oxidation.

The vacuolar degeneration of the liver parenchymal cells has been described before and attributed to hypoxia. Büchner (2) reviewed this finding and found that it was not only common in hypoxic hypoxia but also in static and histotoxic hypoxia. Our findings were consistent with these except for the presence of these changes in 1 dog sacrificed 13 days after decompression. It is unlikely that this was incurred as a result of the experiment, as this would have shown evidence of resolution or regeneration. The most tenable conclusion is that a hypoxia episode was suffered at the time of sacrifice.

The brains examined microscopically were from selected animals sacrificed at various time intervals from 6 days onward following decompression. In only 1 of the animals were lesions

found in the brain. Although the lesions were considered to be residua of prior brain edema, they were so few that they were of academic interest only.

No hemorrhages were found in the brain or in its envelopes. Under conditions of "explosivè" decompression in dogs, with return to ground level within a minute, hemorrhages have frequently been encountered in the wall of the ventricular system and subdurally in the region of the superior sagittal sinus. They have been attributed to the transmission of a pressure wave from thorax to the cerebrospinal fluid, thence to the vetricles and cranium (5). The lack of hemorrhages in our animals might be related to the slower rate of decompression (1 second versus 0.012 to 0.036 second) and to the smaller change in pressure (approximately 180 mm. Hg). Hemorrhages have also been found in the brain substance of animals explosively decompressed and then recompressed either immediately or after a period of 2.5 or 7 minutes. Recompression under all 3 conditions was carried out in 1 minute. When recompression was slow, hemorrhages were few, suggesting that the occurrence of hemorrhages was directly related to speed of recompression (4). Examination of the spinal cord in 5 of our animals revealed tiny diapedetic hemorrhages in 2, mainly in the gray matter. These were thought to have occurred agonally and, thus, were presumably incidental.

One dog of the series became paralyzed. This animal was 1 of a group of 6 which were decompressed to "vacuum" in 1 second, held there for 120 seconds, then recompressed to ground level in 30 seconds. Denitrogenation was not done prior to the chamber run. On decompression, unconsciousness occurred in this particular animal at about the same time as in the other 5. The only difference noted on recovery was the prolonged blindness that this dog manifested. There was no anatomic explanation for this. The precise time at which paralysis developed in this animal is not known, but when it was examined 24 hours after exposure, it was paralyzed. Autopsy performed 87 days later revealed no peculiarities of the lungs or heart. For example, there was no myocardial-septal defect, nor was there an excess of adipose tissue. None of the other 5 dogs in this group showed any unusual clinical manifestations. Autopsies were performed on 3 of them. The brain was examined in 2 and the spinal cord in 1, but neither the brains nor the spinal cord showed changes. Thus, there was no clue as to why 1 particular dog in this group of 6 became paralyzed.

The lesions in the spinal cord were limited to the white matter, as is frequently the case in decompression sickness with spinal involvement (7, 8, 9). The observation that the lesions were randomly distributed and that, at some levels, these were virtually limited to one side of the cord, strongly suggests gas-bubble embolization as the chief basis. This view is supported by the radial disposition of some of the lesions in the lateral columns that were oriented to the course of vessels (fig. 7). Were autochthonous bubble formation in the cord the chief, or even a significant, pathogenic factor, a much more equal distribution of the lesions on the two sides of the cord would have been expected. This is not to say that autochthonous bubble formation in the cord did not occur. Hypoxemia per se could not have induced the cord lesions, as it is well known that the cord is far less sensitive than the brain to a lack of oxygen (10). In this animal, the brain appeared normal. It is also highly unlikely that air emboli, resulting from the rupture of alveoli, could have been instrumental, as under these conditions, the brain would be the chief site of lodgement of the emboli.

The question then arises as to the source of gas bubbles presumed to have been present in the bloodstream. Pertinent observations are available from the literature. During the course of more than 700 explosive decompressions of laboratory animals to simulated altitudes up to 50,000 feet (87 mm. Hg), with decompression carried out almost immediately thereafter, no evidence of bubble formation in the bloodstream, in the cerebrospinal fluid, or elsewhere was observed (14). Further experiments were carried out under much the same decompression conditions (decompression in 0.56 to 0.59 second to a simulated 43,000 to

50,000 feet—122 to 87 mm. Hg), except that the animals, which included 1 dog, 2 cats, and 19 guinea pigs were kept at altitude for periods up to 60 minutes. At the moment of decompression, the chamber was flooded with oxygen and the oxygen flow was maintained. The dog and the 2 cats survived the 60-minute exposure, and no bubbles were found. The guinea pigs were more sensitive; 13 died at time periods varying from 4 to 15 minutes after decompression. Of the 13, gas bubbles were found intravascularly in 7, mainly in the veins of the abdomen and in the inferior vena cava, and occasionally in the right side of the heart. In the 6 guinea pigs which survived the 60-minute run, no bubbles were observed. In the animals that died, the bubbles were thought to have been generated after death (15).

More pertinent to our problem are the studies on dogs explosively decompressed to a simulated altitude of 70,000 feet (30 mm. Hg), for under these conditions, vaporization of body fluids occurs, as in our animals. (Vaporization of the fluids in dogs occurs at 47 mm. Hg at a normal body temperature of 37° C.) In one study, in dogs, of the effects of decompression to 72,000 feet (30.5 mm. Hg in 0.150 second), x-rays of the thorax were made at 5, 30, 60, and 90 seconds. Then the animals were recompressed to ground level. At 30 seconds, in addition to vapothorax and other abnormalities, there was a suggestion of gas in the right auricle of the heart. At 60 seconds, gas was definitely present in the right auricle, and at 90 seconds in both right and left heart. Control animals decompressed to 50,000 feet (87 mm. Hg) showed no such changes. Autopsy of the animals decompressed to 72,000 feet revealed considerable dilation of the heart, more on the right side than on the left. No mention was made of the presence or absence of gas bubbles in the bloodstream (13).

In a further study (12) in which dogs were decompressed to 70,000 feet (30 mm. Hg) in 0.02 second, the problem of gas bubbles in the blood stream was dealt with. Blood was withdrawn through glass cannulae which were placed in the carotid artery and external jugular vein and from catheters placed in the heart and aorta. Gas bubbles were also sought in vessels in regions of the leg from which the skin had been removed, and they were also sought at autopsy. In practically all the dogs studied, macroscopic gas bubbles issued from the vein, the artery, or from both in 5 to 45 seconds following the decompression. Bubbles were also observed in samples of heart blood. In the exposed region of the leg, macroscopic gas bubbles were noted in small arteries within 30 seconds after decompression and within 180 seconds in veins. At autopsy, small bubbles were occasionally seen in the heart or in adjacent large vessels. It was postulated that cavitation from rapid changes in pressure and the turbulence in or near the heart might have accentuated bubble formation. The bubbles appeared to form in the heart and pulmonary vessels, where turbulence is considerable, and at the bifurcation of arteries, blocking the circulation (12).

In viewing the heart through a chest window following decompression to 70,000 feet (30 mm. Hg), gas bubbles were noted in the coronary arteries in 30 seconds. X-rays also revealed gas in the right heart in 30 seconds and in the left heart in 1 minute (3).

Thus, ample data indicate that gas bubbles circulate in the bloodstream under conditions in which vaporization of body fluids occurs. Presumably, the bubbles are initially composed of water vapor, then take up other gases available in the bloodstream. The opinion that the lesions of the spinal cord in our paralyzed dog were due chiefly to gas-bubble embolization appears borne out by previous studies (3, 12, 13, 15). This animal was exposed to "vacuum" for 120 seconds. Recompression was comparatively slow—namely, in 30 seconds. This relatively long exposure to "vacuum" is probably an important factor in the generation of sufficient bubbles to embolize the spinal cord. Why this particular dog was the only one that became paralyzed remains unexplained.

The present data seem to indicate that exposure to "vacuum" for 120 seconds following explosive decompression, with a total of 150 seconds at lowered barometric pressure, may be near threshold for disabling spinal cord damage.

It may be concluded from these experiments that the morbidity incurred from exposure to atmospheric pressures less than 2 mm. Hg (150,000 feet) will primarily be proportional to the duration of anoxia if the time of decompression is not less than 0.2 second. If the exposure time exceeds 120 seconds, a significant number of deaths will occur. If the exposure is less than 90 seconds, however, only minor, transient changes may be anticipated. It is possible that permanent neurologic damage can occur if the exposure is sufficient to cause gas embolization. The relevance and application of this to future space hazards is self-evident, but the gap between possible anatomic damage and functional neurologic damage becomes more self-evident. Therefore, more study is needed on the effects of this type of insult on the functional operation of the central nervous system.

REFERENCES

1. Booth, M. Fragility of erythrocytes as affected by anoxia, CO_2 inhalation and insulin hyperglycemia. Proc. Soc. Exp. Biol. Med. 46:640 (1941).

2. Büchner, F. Pathology of general oxygen deficiency. *In* German Aviation World War II, vol. I, ch. IV-O, pp. 414-425. Washington, D.C.: U.S. Government Printing Office, 1950.

3. Burch, B. H., J. P. Kemph, E. G. Vail, S. A. Frye, and F. A. Hitchcock. Some effects of explosive decompression to 30 mm. Hg upon the hearts of dogs. J. Aviation Med. 23:159 (1952).

4. Cole, C. R., D. M. Chamberlain, B. H. Burch, J. P. Kemph, and F. A. Hitchcock. Pathological effects of explosive decompression to 30 mm. Hg. J. Appl. Physiol. 6:96 (1953).

5. Edelmann, A., W. V. Whitehorn, A. Lein, and F. A. Hitchcock. Pathological lesions produced by explosive decompression. J. Aviation Med. 17:596 (1946).

6. Greely, P. O., J. P. Baumberger, and W. E. Beng. Experiments in explosive decompressions. Final Report CAM OEM CMR 258, 27 Dec. 1943.

7. Haymaker, W. Decompression sickness. *In* Lubarsch, O., F. Henke, and R. Rössle (eds.). Handbuch der Speziellen Pathologischen Anatomie and Histologie, vol. 13, part 1, pp. 1600-1672. Berlin: Springer, 1957.

8. Haymaker, W., and A. D. Johnston. Pathology of decompression sickness. A comparison of the lesions in airmen with those in caisson workers and divers. Milit. Med. 117:285 (1955).

9. Haymaker, W., A. D. Johnston, and V. M. Downey. Fatal decompression sickness incurred in the course of jet plane flights. A clinicopathological study of two cases. J. Aviation Med. 27:2 (1956).

10. Haymaker, W., and H. Strughold. Atmospheric hypoxidosis. *In* Lubarsch, O., F. Henke, and R. Rossle (eds.). Handbuch der Speziellen Pathologischen Anatomie und Histologie, vol. 13, part 1, pp. 1673-1711. Berlin: Springer, 1957.

11. Hitchcock, F. A. Studies in explosive decompression. Physiological and pathological effects. WADC-TR 53-191, Dec. 1953.

12. Kemph, J. P., and F. A. Hitchcock. Changes in blood and circulation of dogs following explosive decompression to low barometric pressures. Amer. J. Physiol. 168:592 (1952).

13. Vail, E. G. Dynamic responses within the thorax to explosive decompression. *In* Hitchcock, F. A. (ed.). WADC-TR 53-191, pp. 55-85, Dec. 1953.

14. Whitehorn, W. V., A. Lein, A. Edelmann, and F. A. Hitchcock. The effect of explosive decompression on cerebrospinal fluid pressure. Amer. J. Physiol. 148:253 (1946).

15. Whitehorn, W. V., A. Lein, and F. A. Hitchcock. The effect of explosive decompression on the occurrence of intravascular bubbles. J. Aviation Med. 18:392 (1947).

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Task No. 775801	9b. OTHER REPORT NO(S) *(Any other numbers that may be assigned this report)*
d.	

10. AVAILABILITY/LIMITATION NOTICES
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11. SUPPLEMENTARY NOTES	12. SPONSORING MILITARY ACTIVITY
	USAF School of Aerospace Medicine Aerospace Medical Division (AFSC) Brooks Air Force Base, Texas

13. ABSTRACT

To estimate the times of consciousness, collapse, and survival of animals exposed to near-vacuum environments, 126 conscious dogs were rapidly decompressed in either 1 or 0.2 second from 35,000 feet, while breathing oxygen, to a pressure less than 2 mm. Hg absolute. Groups of 6 dogs each were exposed to this low pressure for periods of time ranging from 5 to 180 seconds, with and without prior denitrogenation, and then recompressed to 35,000 feet with oxygen in either 5 or 30 seconds. The dogs collapsed within 9 to 10 seconds after decompression, as determined from motion picture films. Simultaneously, the effects of anoxia, water vapor, and other evolved gases were apparent, resulting in a generalized muscle spasticity, a few gasps, momentary convulsive seizures, apnea, and gross swelling of the body and extremities. All dogs exposed for less than 120 seconds survived, despite evidence of lung involvement. Respiration recommenced spontaneously either during recompression or at ground level, provided the heart was beating; otherwise, death was inevitable. The longer the exposure time, the more prolonged was the time for recovery which usually ranged from a few minutes to a few hours, except for 1 dog which exhibited a severe postdecompression paralysis with gradual recovery over a period of several weeks. Exposures of 120 to 180 seconds resulted in approximately 15% to more than 80% fatalities, respectively. Denitrogenated dogs tended to show a slightly better survival rate. As might be expected, the shorter the exposure time and the faster the recompression rate with oxygen, the better were the chances for uneventful and prompt recovery.

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Unclassified

Security Classification

14. KEY WORDS	LINK A		LINK B		LINK C	
	ROLE	WT	ROLE	WT	ROLE	WT
Stress physiology						
Decompression, rapid						
Vacuum, survival and mortality in						
Consciousness, time of in vacuum						
Anoxia						
Ebullism						

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DOCUMENT CONTROL DATA - R&D

(Security classification of title, body of abstract and indexing annotation must be entered when the overall report is classified)

1. ORIGINATING ACTIVITY *(Corporate author)*
USAF School of Aerospace Medicine
Aerospace Medical Division (AFSC)
Brooks Air Force Base, Texas

2a. REPORT SECURITY CLASSIFICATION
Unclassified

2b. GROUP

3. REPORT TITLE
EXPERIMENTAL ANIMAL DECOMPRESSIONS TO LESS THAN 2 mm. Hg ABSOLUTE (PATHOLOGIC EFFECTS)

4. DESCRIPTIVE NOTES *(Type of report and inclusive dates)*
Apr. 63 - Apr. 65

5. AUTHOR(S) *(Last name, first name, initial)*
Dunn, James E., II, Captain, USAF, MC
Bancroft, Richard W.
Haymaker, Webb
Foft, John W., Captain, USAF, MC

6. REPORT DATE	7a. TOTAL NO. OF PAGES	7b. NO. OF REFS
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c. Task No. 775801	9b. OTHER REPORT NO(S) *(Any other numbers that may be assigned this report)*
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11. SUPPLEMENTARY NOTES	12. SPONSORING MILITARY ACTIVITY
	USAF School of Aerospace Medicine Aerospace Medical Division (AFSC) Brooks Air Force Base, Texas

13. ABSTRACT Pathologic examination of tissues of dogs rapidly decompressed to less than 2 mm. Hg absolute was performed. Of the 126 dogs decompressed, 92 were autopsied at 3 time intervals: within 30 minutes, 2 to 5 days, and 1 to 3 weeks post-decompression. Gross examination of the tissues was performed on all autopsied animals. Lung damage was graded 1+ to 4+ according to the amount of edema, emphysema, atelectasis, and hemorrhage present alone or in combination. Microscopic examination of the tissues was performed on selected dogs from the various groups.

The most impressive finding was the absence of major pathologic damage, except in the lungs, unless the exposure time exceeded 120 seconds. By varying time of decompression and time of exposure to less than 2 mm. Hg, it was possible to separate the pathologic effects of anoxia versus time of decompression. In all dogs, the severity of lung damage increased with duration of the anoxic exposure. In groups with comparable exposure times, the dogs decompressed in 1 second exhibited pulmonary congestion, edema, and hemorrhage, while those decompressed in 0.2 second showed predominantly more petechiae and emphysematous changes. Denitrogenation appeared to reduce the incidence and severity of the lung damage. Animals autopsied at the later postdecompression periods showed evidence of resolution of all lesions, especially in the lungs. For the exposures that were longer than 120 seconds, gross examination of other organs and tissues showed increasing congestion and some hemorrhage. The brains showed engorgement without evidence of hemorrhage. One dog that was paralyzed from the exposure had numerous demyelinated lesions of the spinal cord that seemed to be the result of gas bubble emboli.

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Security Classification

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